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Impartial Medical Testimony

The Medical Expert Testimony Project of the New York Bar

This monograph* records one of the most remarkable medico-legal experiments ever undertaken, prompted by

'... the uncertainty, confusion, and waste of time resulting from the presentation of widely conflicting medical opinion evidence by partisan doctors retained by the respective parties. Such evidence, which occupies a large part of the trial time in any personal injury action and which is necessarily of major importance in pre-trial settlements, is too often colored and calculated to persuade a court or jury to a partisan point of view, rather than to present an objective medical analysis and appraisal. Instead of being aided by such expert testimony the triers of the facts are confounded and confused, and left without reliable guidance in choosing between diametrically opposed medical opinions.

Basically, the idea behind the Medical Expert Testimony Project was to set up panels of neutral, outstanding physicians in various specialized branches of medicine. These experts would be available at the call of the court to make medical examinations of plaintiffs in personal injury cases, report their findings, and, if necessary, testify, in those cases in which the medical aspects were controversial and substantial.' (p. 3).

* *Impartial Medical Testimony: A Report by a Special Committee of The Association of the Bar of the City of New York on the Medical Expert Testimony Project.* (1956. Pp. 188, $3.95). New York: The Macmillan Company.

Doctors for the expert panel were selected by a joint Committee of the New York Academy of Medicine and the New York County Medical Society, to ensure members of the highest qualification and standing, appointed solely on a professional basis.

After operating for 2 years, the experiment was judged a success mainly for the following reasons:

'1. The Project has improved the process of finding medical facts in litigated cases.

2. It has helped to relieve court congestion.

3. It has had a wholesome prophylactic effect upon the formulation and presentation of medical testimony in court.

4. It has proved that the modest expenditure involved effects a large saving and economy in court operations.

5. It has pointed the way to better diagnosis in the field of traumatic medicine. Unlike the others listed above, this accomplishment is an unexpected dividend, which was not in contemplation when the Project was initiated.' (p. 5).

A private grant of $40,000 made the investigation possible in the first place, but as a result of this pilot experiment, the scheme has been adopted as a routine procedure in the Supreme Court of the State of New York in the First Department. The expense of operating the plan is now included in the court budget approved by the Board of Estimate of the City of New York.

In principle the panel is reminiscent of the Danish Medico-Legal Council to which written questions may be submitted, and from which written answers (not binding on the court) are obtained. It is even theoretically possible for the members of the Medico-Legal Council to be brought into the witness-box for cross-examination.

The New York experiment revealed a far more flexible process, better adapted to the volume of work which had to be coped with. Its positive achievements are very considerable and clearly very admirable. They indicate that the scheme has much to commend it as a model to be studied closely in other parts infested with the unprincipled hireling medical witness.

Although the Project may have remedied an appalling local situation created largely by certain types of medical practitioner, it would be wrong to indict, by implication, medical witnesses as a class. The New York experiment will succeed in liquidating the evil of the witness, the quality of whose evidence is determined by the quantity of his hire; but in many countries the corrupt, partisan witness does not flourish.

Certain serious problems remain to be faced in implementing the New York Project elsewhere. The considerable cost of the undertaking must increase *pari passu* with its general adoption and extension. Who will be responsible for what may become a vast annual expenditure?

A disturbing feature of the system is that the testimony of those chosen for the panel must necessarily, especially in the course of time, acquire an

impeccable quality—and this is undesirable no matter how eminent the witness may be in his own professional sphere. Panel members may acquire (and not of their own seeking) a myth of infallibility, so that their opinions become virtually like those of an unchallengeable Spilsbury, whose *ipse dixits* will decide an issue—a procedure which may dispose of a case without necessarily dispensing justice in the process. The unwarranted additional authority which the panel experts may acquire can, however, probably be countered by ensuring a fairly frequent rotation of members entitled to serve, so that no practitioner can be entrenched for prolonged periods of time.

Opinion in medical and scientific matters is never static and there may be suitable practitioners who will not qualify for admission to a panel because of their physical youth, or their unpopularity for reasons unconnected with their professional ability, although they may be in a better position to guide a court than some of their colleagues. The system may also, in some countries, permit the vagaries of medical politics to decide appointments which should be unconnected with medico-political intrigue.

Because of the stature which a panel expert must acquire, merely by virtue of being on the panel, that potent organ for detecting the truth, viz. cross-examination, may well be endangered (if not emasculated) and medical issues may drift into a pattern of 'trial by Harley Street', a possibility which has certainly evoked very little enthusiasm in legal circles in the United Kingdom.

The principles involved in employing disinterested medical experts as witnesses is by no means new. In many countries the State provides and pays for just such witnesses in criminal trials. Everything is arranged to diminish the possible intrusion of partisan evidence by such experts, who conscientiously carry out their difficult duties to the best of their abilities and actuated by the highest principles and ethical motives. Yet even such witnesses can be in error, may have to make retractions and do not always succeed in convincing the courts of the correctness of their hypotheses, especially when they are subjected to a cross-examination based on an adequate briefing of counsel.

We should thus not lightly discard or modify sound and well-tried principles, tested out over the centuries, which govern the admission of expert testimony. We must not lightly abandon the security enshrined for litigants and accused persons in the current rules of court and in the mighty weapon of cross-examination.

If, in any state or country, only one professional witness were cautioned, reprimanded or removed from the statutory register entitling him to practise his calling, because of his unethical conduct, much would be achieved (and rapidly and inexpensively) without the danger of creating panels of untouchable medical witnesses, each invested with a sacrosanct halo of testimonial capacity.

Taylor's Principles and Practice of Medical Jurisprudence

A New Edition

The impressive quality and the encyclopaedic scope of *Taylor's Principles and Practice of Medical Jurisprudence** are reflected once again in this new (eleventh) edition of a reference work which has become a *sine qua non* for all medico-legal practitioners. For some 9 decades *Taylor* has mirrored advances in knowledge and practice in this field, as they justified their inclusion in textbooks; and the wealth of experience enshrined in its pages by a succession of distinguished editors (who have also been outstanding forensic practitioners) has established its pre-eminence as a guide and an authority.

On this occasion Sir Sydney Smith (editor of this work since 1928) has been assisted by Dr. Keith Simpson in revising the first volume.† Dr. Simpson's choice is a happy one, since it marks once again an appropriate association of Guy's Hospital with a volume written in the first place by Dr. A. S. Taylor when he was Lecturer in Medical Jurisprudence at Guy's Hospital Medical School—a position he held for 46 years.

The substantial changes in the new edition are summarized by Sir Sydney Smith in the Preface:

'. . . a somewhat more radical revision has been attempted in order to keep *Taylor* abreast of the times. Several sections—notably those on Post-Mortem Changes; on Intersexuality as an identity problem; on the general procedure of Criminal Investigation; on Regional Wounds; Blood in Identity; Trauma and Disease—and the sections on Asphyxia and Life Assurance have been largely rewritten'. (p. v).

The revised section on *Asphyxia* incorporates an entirely new exposition of this condition, in so far as it results from mechanical interference with respiration. The pioneer work of Gordon drew attention to the distinction which must be made between the general, non-specific signs throughout the body due to oxygen lack (however caused), and the specific signs due to the manner in which the asphyxial oxygen lack was initiated. In throttling or strangling, e.g. there may be small haemorrhages in or on the surfaces of internal organs. These occur in many non-asphyxial conditions and are therefore general signs, not diagnostic of asphyxia (in the sense used here as due to mechanical interference). The local marks on and in the neck (produced by the strangling ligature or the throttling fingers) provide the only diagnostically important evidence on which, *inter alia*, a diagnosis of

* *Taylor's Principles and Practice of Medical Jurisprudence.* Edited by Sir Sydney Smith, C.B.E., LL.D., M.D. (Edin.), F.R.C.P. (Edin.), D.P.H., F.R.S. (Edin.), assisted by Keith Simpson, M.D., Lond.(Path.). (Pp. 609 plus Index. With 1 Coloured Plate and 60 text-figures. 70s.). 11th ed., 1956. London: J. & A. Churchill Ltd.

† Volume 2 will be issued towards the end of this year.

asphyxia may rest. *Taylor* now recognizes and expounds this basic distinction which disposes of asphyxia as a pathological entity which can be recognized from the autopsy signs in the viscera alone. The persistent fluidity of the blood after death and the much reported Tardieu spots are no longer regarded as pathognomonic of asphyxia (pp. 456, 458). These myths have been exposed; but the special importance that small haemorrhages may have for the diagnosis of asphyxia, when they are confined to regions in which the venous return has been obstructed, is not neglected.

An instructive case is illustrated (Fig. 51, p. 457) in which intense cyanosis of the face, with haemorrhages on the brow and the eyelids, was at first diagnosed as due to (mechanical) strangulation. In fact, death was due to aspirin poisoning. This case epitomizes the totally new approach advocated in the study of the post-mortem problem involved in the diagnosis of asphyxia when only cadaveric evidence is available.

The more recent work by Gordon *et al.* (published in this Journal, 1955, **2**, 31) demonstrates equally forcefully the hazards inherent in relying unduly on such signs as internal petechial haemorrhages, since these workers have shown that the typical Tardieu spots can occur spontaneously after death.

The care bestowed on the re-writing of the section on *Asphyxia* is characteristic of the way in which the rest of the volume has been handled. Forensic practitioners throughout the world will therefore welcome the first volume of this authoritative work and look forward to the completion of the revision with the publication of the second volume later this year.

BOOK REVIEW

Polson's Forensic Medicine

The Essentials of Forensic Medicine. By C. J. Polson, M.D., F.R.C.P. (1956. Pp. x + 561. 30s.). London: English Universities Press Limited.

Professor Polson holds the Chair of Forensic Medicine at the University of Leeds. His volume is 'an amplification of lectures delivered in the University . . . and it embodies experience gained through participation in a pathology service for the Home Office and for certain H.M. Coroners in Yorkshire' (p. v).

Professor Polson's contribution to the growing list of textbooks issuing from the principal forensic teaching centres in the United Kingdom is unusual in that it deliberately discards illustrations and classifications. The text, however, is punctuated by a very considerable number of case histories and records which exemplify many points of practical importance. The bibliography is comprehensive and indicates the author's scholarly approach.

The condensation of text is not without risk. No attention, e.g. is drawn to the need for meticulous controls in determining the origin of blood (e.g. whether it is human) and none of the limitations currently recognized are mentioned, e.g. that current laboratory procedures establish blood as primate and not human. The same lack of attention to controls applies to the account of the principles involved in the grouping of blood stains. As Professor Polson's volume is intended also for barristers, police officers, etc., these important features should be included. They are also necessary for an appreciation by undergraduate students of the exacting demands made upon those who undertake to give expert testimony.

The omission of a section on toxicology is commendable. The clinical features of poisoning do not merit any special account in a textbook of forensic medicine. The practitioner is thoroughly trained to deal with these problems as a result of his general knowledge of medicine.

Professor Polson's volume is an interesting and useful addition to the medico-legal library.

Suicide with Natural Gas‡

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and

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The public utility company in Baltimore, Maryland, converted from manufactured illuminating gas to natural gas during 1950. By the middle of September of that year the entire city had been converted. This was dramatically reflected in the statistics of the Medical Examiner's Office which showed a drop in deaths due to illuminating gas to zero level during the period from September 1950 to 16 September 1952. In the corresponding 2-year period preceding September 1950, there had been 61 deaths, most of them suicidal. This change had been so startling that it was the subject of editorial comment in the local press on the mornings of 10 and 16 September 1952, wherein it was pointed out that death could not be caused by the natural gas which was currently in use. Whether by coincidence or as an answer to the fancied challenge set forth in the editorials, there was reported to the Medical Examiner's Office on 16 September 1952 the following set of circumstances.

At approximately 5.45 p.m. on 16 September 1952, the police were called to a home in Baltimore by a woman who stated that when she had arrived home at about 5.40 p.m. she had noticed the strong odour of illuminating gas issuing from the second floor of the building. Upon investigation the police found the body of the woman's husband, a 37-year-old white man. The body was on the floor next to a gas pipe. Two pipe wrenches were found near the body. The cap of the gas pipe had been removed and the open end of the pipe, from which gas was flowing, had been placed under

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‡ Read at the Fifth Annual Meeting, American Academy of Forensic Sciences, 26 February 1953.

a plastic raincoat which completely covered the head and shoulders of the deceased. The door and windows of the room had been sealed but not locked.

The wife of the dead man stated that her husband had told her a week before his death that she would be better off without him and that on 6 September 1952 he had locked himself in a clothes closet and had taken some sedative pills, which, however, had no ill effect. He had been despondent for some time and had not worked for about 9 weeks before his death. It was also stated that he had attempted suicide with illuminating gas about 4 years earlier.

Autopsy was performed approximately 16 hours after discovery of the body and showed no gross changes except in the lungs, which were congested and edematous. A sample of spinal fluid was drawn for alcohol determination and showed 0.06% ethyl alcohol. Analysis of the blood for barbiturates was negative. Blood was collected from the heart under oil, for analysis for gases. Since it was known that the gas company on occasion would add manufactured gas to the natural gas, a carbon monoxide determination was made by the method of Gettler and Freimuth.[1] This showed a carbon monoxide hemoglobin saturation of 5%, which is within normal limits. A further check with the gas company was made and we were informed that no manufactured gas had been in the mains during the period covered by this case.

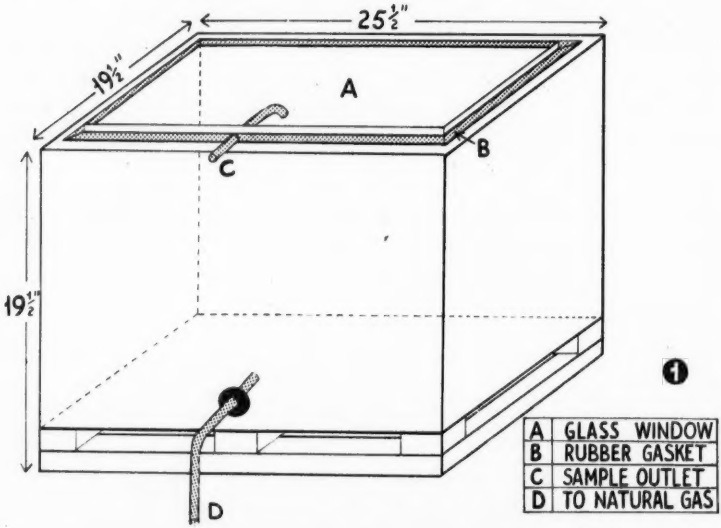
Further analysis of the blood was made for its oxygen content by the method of Van Slyke and Neill.[2] This showed an oxygen content of 6.0 vol. per cent. as compared with a capacity of 21.2 vol. per cent. for the same sample. There was no indication found of any solubility of the hydrocarbons in the natural gas in the blood.

These results indicated that the cause of death was primarily anoxic in nature caused solely by a decrease in available oxygen brought about by displacement of the air by natural gas. It has always been assumed that this would be the only way by which natural gas could cause death. However, a search of the literature discloses a remarkable scarcity of direct evidence for this conclusion, only 2 papers dealing with the physiological effects of natural gas being found.[3, 4] Accordingly, a series of experiments with monkeys was carried out.

A large chamber was arranged as shown in Figs. 1, 2. Each of 4 monkeys (weighing between 5¾ and 6½ lb.) was then used as a subject for exposure to the illuminating gas coming directly from the tap in the laboratory.* Before starting each experiment, a sample of blood was collected under oil by heart puncture. When the monkeys reached unconsciousness, a sample of gas was obtained from inside the chamber by displacement of water. A

* The analysis of this gas showed the following composition: carbon dioxide, 0.4%; methane, 94.8%; ethane, 3.2%; propane, 0.6%; butane and higher hydrocarbons, 0.4%; non-condensable gases, 0.6%.

second gas sample was obtained from the chamber when the animals died. After death the animals were immediately autopsied and a sample of blood was again collected under oil by heart puncture. All the monkeys, except



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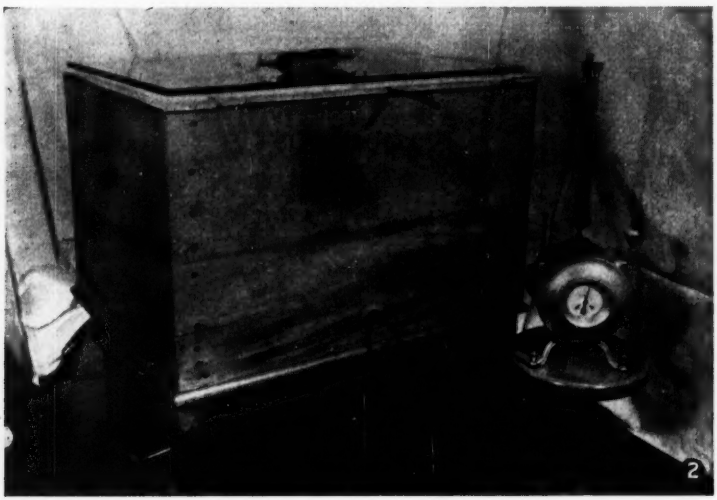

monkey #4, were kept in the gas chamber until death. The latter animal was removed from the chamber after he had become unconscious. In a

period of 4 minutes he had regained full consciousness and was just as lively as he had been at the start of the experiment.

Each of the blood samples was analyzed for carbon dioxide content and oxygen content. The results of these analyses are shown in Table I.

Table I

Animal #	*Time of Exposure*	*Carbon Dioxide Content Volumes %*		*Oxygen Content Volumes %*	
		Normal	*Post Mortem*	*Normal*	*Post Mortem*
1	1 hr. 13 min.	31·5	17·1	9·3	2·2
2	1 hr. 19 min.	24·2	13·7	10·7	1·8
3	1 hr. 23 min.	26·1	17·6	10·2	3·1
*4	1 hr. 14 min.	31·3	27·0	9·9	4·4

* Monkey #4 was removed immediately upon becoming unconscious and revived while blood sample was being obtained.

Each sample was analyzed for dissolved natural gas by determining the alcohol soluble fraction present in the mixed gases liberated from the blood. No dissolved natural gas was found.

The results of the analyses of the gas samples obtained during the experiments are shown in Table II.

Table II

	Sample #1 (Animal Unconscious)		*Sample #2 (Animal Dead)*	
Animal #	*CO_2 (Vol. %)*	*O_2 (Vol. %)*	*CO_2 (Vol. %)*	*O_2 (Vol. %)*
1	0·7	6·0	0·7	4·2
2	0·9	7·5	1·0	3·9
3	0·9	5·2	0·8	3·7
4	0·9	5·0	—	—

The balance of the gas in the chamber in each of the above analyses was natural gas.

Eighteen days after the exposure to the natural gas, monkey # 4 was sealed into the chamber and allowed to remain there until he had exhausted the supply of oxygen in the chamber and thus expired. This process required 11½ hours. A sample of blood was collected under oil by heart puncture before the animal was placed in the chamber and a similar sample was obtained at death. These were analyzed for CO_2 content and O_2 content with the results shown in Table III.

Table III

	Normal	*Post Mortem*
CO_2 content (Vol. %)	30.5	21.2
O_2 content (Vol. %)	10.1	2.0

Analysis of samples of air taken from the chamber when the monkey was unconscious and later when he was dead gave the results shown in Table IV.

Table IV

	Volumes % CO_2	*Volumes % O_2*
Monkey unconscious	8.4	11.2
Monkey dead	8.9	10.7

All of the above results are in keeping with results reported in the other types of anoxic deaths.[5–7]

The symptoms exhibited by the animals were quite uniform. They first became sleepy, then toppled over and began to breathe heavily. They then lapsed into unconsciousness and showed the typical muscular twitching associated with asphyxia, with death following shortly thereafter.

Summary and Conclusions

A case of suicidal asphyxiation through the use of natural gas is reported.

Experimental studies with monkeys show that the sole effect of the natural gas is the exclusion of oxygen thus causing anoxic death.

There is no specific effect of the components of the natural gas.

We wish to express our thanks to Dr. E. W. Guernsey, Director of Research of the Consolidated Gas, Electric Light & Power Co. of Baltimore, for the analyses of the gas samples reported herein.

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The Study of Human Hairs

As an Aid to the Investigation of Crime

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Despite the progress made in the last 25 years in using scientific knowledge to investigate crime, there has been no significant change in the expert's approach to the question of hair identification. Assertions are sometimes made regarding the race, age, sex, occupation and even habitat of a person from an examination of a single hair which show either a superhuman knowledge of the subject or complete ignorance of it.

Certainly it is possible, in a majority of cases, to identify a hair as being of human origin and, generally, to say from which part of the body it has come. Microscopic examination of a cross-section of the hair may show certain racial characteristics. The lank hair of the oriental is round or near-round in cross-section. The curly frizz of the African negro may be almost ribbon-like, showing a flattened elliptical cross-section. The hair of the white races exhibits a range of variation between these two extremes, being on the average somewhat oval in cross-section.

The examination of any dust which may be adhering to the hair can assist the investigator in identifying the nature of the occupation of the person from whom the hair has come, but will rarely, if ever, help to find his or her home.

Microscopic examination of hair can tell little about the age of the person from whom it has come. From youth to old age there is little change in the appearance of the hair, apart from the almost inevitable greying. The degree of greyness, in fact, may provide the only means of estimating age and this may give an error of over 40 years. It is well known that some people, have grey hairs in their early 20's while others possess an unchanged naturally coloured head of hair in their 60's or even later. It is interesting to note in passing that colour and quantity seldom go together. It frequently happens that those who go grey early keep more of their hair as they grow older than those who retain the original colour. If any generalization can be made about age and hair colour (and it is only

in generalizations that one can dogmatize about hair) it is that grey hair becomes more common from the late 30's onwards.

Not so long ago the sexing of hair presented fewer difficulties than at present. The long, uncut hair came almost certainly from a female head and the short cut hair from a male head. Nowadays the reverse may well be true, for the unpredictable dictates of tonsorial fashion do not make the work of the forensic scientist easy. Again, one can only say that, in general, the head hair of the female of the species is finer than the male's. The exceptions, however, may be embarrassingly numerous.

Despite all that the scientific investigator of fiction may say to the contrary, it is not possible (in the present state of knowledge) definitely to identify a sample of hair as having come from one individual's head. The probability of identity may grow with every point of resemblance and with the number of hairs available for comparison, but that probability should never be stated as a certainty. Nevertheless, there is often considerable microscopic difference between heads of hair of similar appearance and colouring; absolute identity between two heads of hair, though theoretically possible, is in practice very rare, if it happens at all. This, of course, applies only to heads of hair and not to the individual hairs which make up those heads. The following case illustrates this point.

Case 1. A Lancashire Naval Establishment was broken into at night shortly after the end of the war. The intruder awakened one of the sleepers and in the struggle which ensued lost a handful of hair before he succeeded in escaping. Samples of hair were obtained by the Naval authorities from all the Naval personnel whose hair was, to the naked eye, of the same colour as, or similar in colour to, that torn from the head of the intruder. The laboratory received about 50 samples of hair, all almost indistinguishable to the naked eye from the one with which they were to be compared. The examination showed, however, that there was no complete agreement between any of them and the original sample, thus removing suspicion from the Naval personnel. Unfortunately, it was not possible to ask the large number of civilians who either lived on or had access to the camp to volunteer for a hair-cut.

When comparing hairs it is usual to make examinations of both the longitudinal and the transverse planes. The principal features to receive attention may be discussed briefly as follows:

Colour: Though there is generally a considerable degree of variation in the colour of the individual hairs in a head of hair, there is still a colour relationship between these hairs. Grey and white hairs are excluded from this colour relationship. It is necessary also to note the distribution of the colour throughout the single hair.

Size and Shape: The length to which a hair will grow depends to some extent on the part of the head in which it is rooted. Around the fringes of the hair, particularly at the temples and the nape of the neck, hairs in both sexes are quite frequently shorter and finer than those in other parts of the head. Tapered hairs, i.e. hairs with uncut pointed ends, may be present in both male and female heads but are certainly rarer, even to-day, in the male than in the female.

Curl: The tendency to curl in hair is exhibited under the microscope as alternate thickening and thinning of the hair along its length (Fig. 1). The extent of this

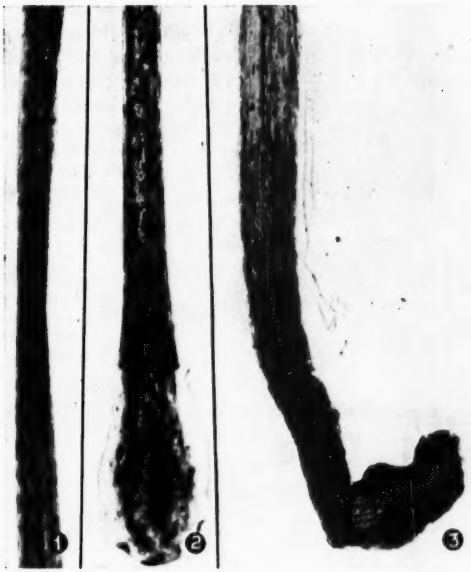

Fig. 1. Curling head hair showing variation in thickness (X 80).

Fig. 2. Root of plucked hair showing healthy bulb and sheath. Note increased detail in region of root (X 100).

Fig. 3. Fallen hair with atrophied root and remnants of sheath (X 100).

thickening and thinning, which is present to some slight degree in most hairs, is the extent of the wave in that hair. This may vary in different parts of the same head. This curling is also characteristic of pubic hairs.

Root: Considerably more detail is generally available for comparison at and about the root of the hair than elsewhere along its length. It may be possible from the examination of the root to tell whether or not the hair has been forcibly removed (Figs. 2, 3). At least some of the specimen hairs required should be plucked.

Medulla: This central structure of the hair can be of considerable value in making hair comparisons. When visible it shows great variation, being more or less continuous, interrupted, central or, rarely, eccentric.

Surface: The surface scales of hairs can provide valuable information. In the case of very young children an examination of the scale structure may give a fairly accurate assessment of age (Figs. 4, 5). Where the hair is dirty, possibly through lack of washing, the dirt adheres more readily around the scale edges, making them stand out more clearly than usual on microscopic examination. The hair surface may also show the effect of disease, as in ringworm or scurf, or it may have adhering to it the eggs of the head louse (Fig. 6).

The variation that occurs between hairs from different parts of the same head makes it preferable and sometimes essential that samples for comparison purposes should be taken from various parts of the head. This variation may, of itself, be of assistance to the examiner as it provides him with additional points for comparison.

Case 2. In December 1950, a grocer's shop near Workington, Cumberland, was broken into and money and goods stolen. Near the point of entry a small comb was found. Some months later this comb, together with samples of hair from the

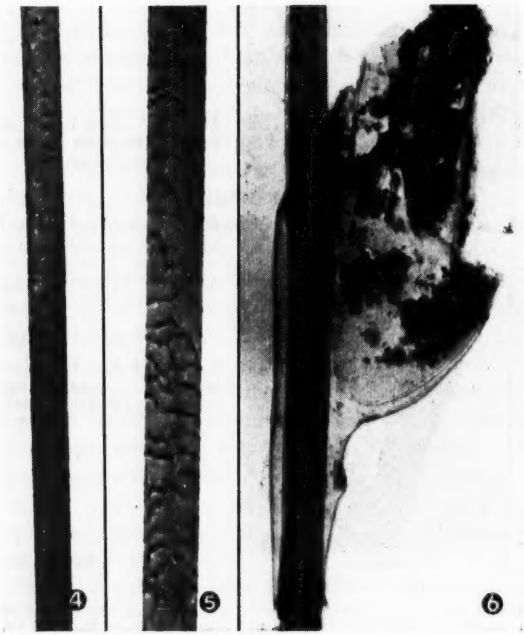

Fig. 4. Cuticular scales of head hair of new-born child. Note smooth scale edges (X 1350).

Fig. 5. Cuticular scales of head hair from an 8 months old baby. Note serrated scale edges (X 1350).

Fig. 6. Egg of head louse hatching (X 100).

heads of 2 men thought to be responsible (one of whom admitted the offence) was examined at the laboratory. Caught in the teeth of the comb were several short cut hairs, 7 in, all, varying in length from 1–9 mm. They all differed slightly in colour and other microscopic characteristics, but they could all have come from the same head. They showed no agreement with the hairs from the head of the men who admitted the offence but each hair was capable of being matched exactly with hairs from the head of the other suspect, who, it was learnt, had had a haircut not long before the offence was committed. (R. *v.* Pooley and McAuley, Carlisle Assizes, 17 May 1951.)

In some cases the presence in a sample of hair of some artificial alteration to its natural state may provide a useful link with another sample. Microscopic examination of the hair will generally reveal whether it has been bleached, dyed, waved or recently cut.

Case 3. Late one night in January 1942, a jeweller's window in Preston, Lancashire, was smashed and a quantity of jewellery stolen. Two men were seen to drive away, one of them dropping a beret while making his escape. When the beret was examined at the laboratory several human head hairs were found inside. The microscope showed them to be dyed brown, their original colour, visible at the roots, being much lighter. Later these hairs were compared with head hairs from a suspect. The hairs from the 2 sources were found to show complete microscopic agreement in both their dyed and their undyed parts. (R. *v.* Thomas, Manchester Assizes, 27 April 1942.)

The appearance of hair under the microscope can be altered by physical treatment other than waving.

Case 4. A woman charged with murdering her husband in their home at Bolton, Lancashire in December 1950, stated in her defence that her husband had quarrelled violently with her, pulled her about by her hair, meanwhile striking her with his fist. To protect herself she had picked up a knife from the table. She claimed that her husband had run on to it when trying to strike her again. A large tuft of hair found on the dead man's left trouser leg was compared with hair from the accused's head. The 2 samples agreed completely in all but one feature; the diameter of the hair from the scene was markedly less than that from the woman's head. It was found, however, that if the hairs from the accused's head were subjected to stretching, the agreement between them and those from the scene became complete (Figs. 7, 8). To provide the required diameter of hair it was necessary to stretch the hair to breaking point; this provided an increase in length of about 50%.

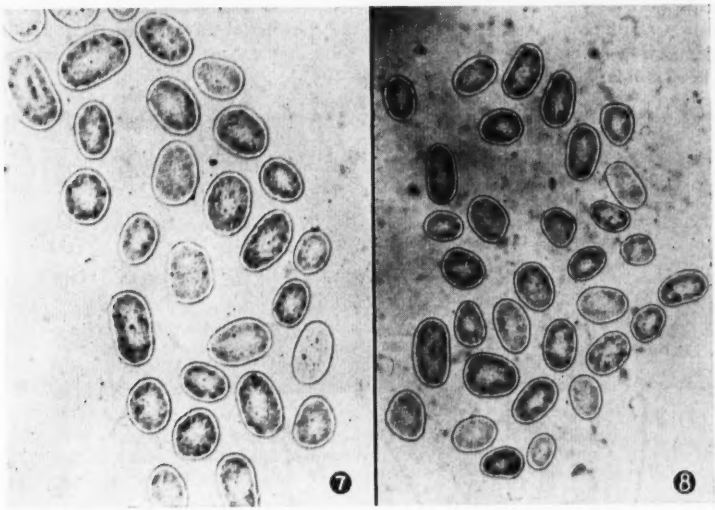

Fig. 7. (Case 4). Transverse section unstretched hairs from accused's head (X 100).
Fig. 8. (Case 4). Transverse section stretched hairs from accused's head. Note decrease in cross-sectional area (X 100).

This was taken as being some corroboration of the accused's story and she was convicted of manslaughter. (R. *v.* Kay, Manchester Assizes, 12 March 1951).

Attempts to repeat this stretching process with hairs from other sources have shown that the extensibility of hairs varies considerably from person to person. In many cases the increase in length has been insufficient to show any appreciable alteration in the diameter of the hair.

In all cases of assault, whether causing injury or not, it has become almost routine to examine the clothing of all persons involved to see whether there has been any interchange of hairs. In indecency cases the search is extended to include pubic hairs and, in cases of indecency

between males, anal hairs also. Evidence resulting from such examinations and the consequent comparisons has frequently provided valuable corroboration in a wide variety of cases of this nature. From time to time cases occur in which the examination by the scientist reveals the presence of hairs from parts of the body other than those already mentioned.

Case 5. After a street brawl in Kendal, Westmorland, a man suffering from head injuries was found lying by the roadside. He was carried to his home where he was found to be dead when examined by a doctor. His injuries, though not severe, included a fractured nose. Internal bleeding from this injury while he had been lying unconscious on the ground had resulted in his death from asphyxiation. The clothing of a man thought to have been concerned in the brawl was later examined at the laboratory. With the exception of the boots his clothing was free from blood but the toe of the right boot was blood stained. Adhering to the blood-stained area was a human head hair and embedded in the blood was a short hair which was identified as a human eyelash. The head hair was compared with hairs from the head, and the eyelash with the eyelashes of the deceased (Figs. 9, 10). Complete agreement was found to exist between the hairs from the two sources. (R. *v.* Tallon, Appleby Assizes, 13 January 1955.)

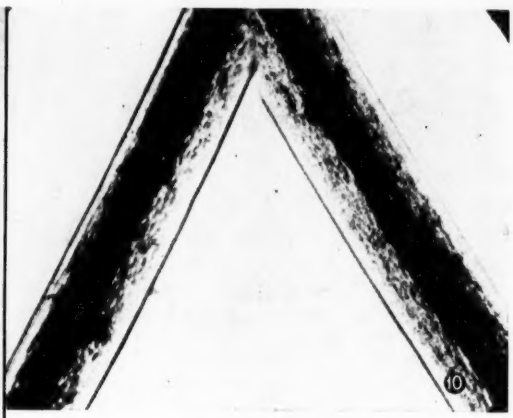

Fig. 9. (Case 5). Eyelash removed from boot. Note the characterististic shape (X 10).

Fig. 10. (Case 5). Comparison between eyelash from boot and eyelash from deceased. Note eccentric medulla and densely pigmented cortex (X 100).

Road accidents involving injuries to one or more persons are from their very nature likely to produce examinations involving hairs from any part of the body, as the following cases illustrate,

Case 6. In October 1944, a road accident occurred at Saltney in Flintshire in which 2 youths were injured. The vehicle involved, which did not stop, was a United States Army truck. Later, with the co-operation of the United States authori-

ties, a truck known to have been in the area of the accident at about the appropriate time was found and examined. On a small nut on the offside mudguard was found a small piece of blood-stained matter. When this was examined at the laboratory it was found to be tissue which gave a positive precipitin reaction for human blood and in which several hairs were rooted (Fig. 11). These hairs were not head hairs. A request was therefore made for hair samples from the injured youths from injured parts other than the head. The hairs in the tissue found on the truck showed complete agreement with hairs in a sample taken from the leg of one of the youths (Fig. 12). (The People of the United States of America v. Mara and Hawke, United States Army General Court Martial, 22 November 1944.)

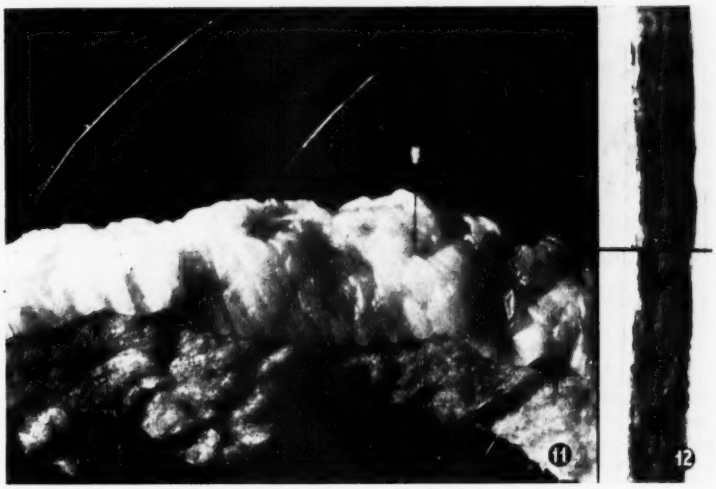

Fig. 11. (Case 6). Flesh found on army truck showing hairs (X 10).
Fig. 12. (Case 6). Comparison between hair from flesh and hair from leg of injured person. Note absence of medulla and dense cortical pigmentation (X 450).

Case 7. A child crossing the road at Preston, Lancashire, was struck and injured by a passing lorry which did not stop. The police investigation showed that no blame could be attached to the lorry driver who, in fact, may well have been unaware that his vehicle had struck the child. Nevertheless, for other reasons, it became necessary to identify the lorry responsible. Later an examination was made of a beer lorry which was known to have passed the place where the accident occurred at about the material time. On a projecting hook on a gantry rail, slung under the offside of the vehicle, was found a fragment of what appeared to be skin. Examination showed it to be skin, rooted in which were several very fine hairs. A request was made for a sample of hairs from the region of the child's main injury —the groin. At first it was maintained that the child had no hairs in that region, but it was pointed out that the hairs were very small and fine, almost invisible to the naked eye, and it was requested that the area of the groin be shaved and the lather sent to the laboratory for examination. In the lather were found numerous very fine hairs with which the hairs found on the lorry were in complete agreement (Fig. 13).

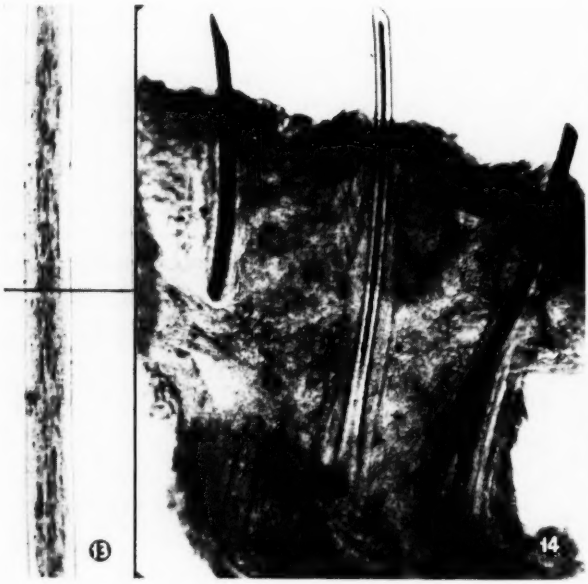

Fig. 13. (Case 7). Comparison between hair found on lorry and hair from groin of injured child. Note absence of medulla and distributed pockets of pigmentation, the fineness of the hair and the scale edges (X 450).

Fig. 14. (Case 8). Flesh found on lorry showing rooted hairs. Note obliquely but sharply cut hair ends typical of shaven hairs (X 25).

Case 8. In March 1945, a Naval rating walking in the gutter of a road at Burscough, Lancashire, facing the oncoming traffic, was struck and injured by a lorry travelling in the same direction. The lorry did not stop. The rating had been walking on the lorry's offside and the lorry had swerved to its offside immediately before the accident. The walker received fairly severe injuries to the left side of his face. Later a motor lorry was examined and from a point on its offside about 5 feet from the ground a fragment of what appeared to be tissue was removed. On microscopic examination this was found to be skin tissue of animal origin and an extract from it gave a positive precipitin reaction for human blood.

Further, it was observed that the skin showed the roots of a number of hairs which had been cut short by some sharp instrument close to the roots (Fig. 14). It was, therefore, a piece of shaved skin such as could have come from a man's face. (R. *v.* Lawson, Ormskirk Magistrates' Court, 4 May 1945.)

In the majority of cases where examinations and comparisons of hairs become necessary, the point at issue is whether or not the hairs examined have come from a certain person. The expert can only say that, as a result of his examination, he is of the opinion that the hairs in question do or do not agree with hairs in various samples submitted to him for comparison purposes. If they do agree with hairs in a certain sample he cannot say that they could not have come from a different person. If, on the other hand, no agreement can be found between 2 samples of hair he may be justified in stating definitely that they are not from the same source. In the last 4 cases quoted the presence of hairs in the positions in which they were found is of itself a matter of some significance. In Case 5 the presence

of both a head hair and an eyelash on the accused's boot makes it probable that a claim that the hairs did not come from the dead man would be unlikely to meet with sympathetic consideration from a jury. This, however, is not the concern of the expert and if asked under these or similar circumstances whether such hairs could have come from another person, he must agree with the possibility. His duty is to present the facts to the best of his ability, stating only what his knowledge allows and his experience supports.

Summary

The identification and comparison of human hairs have been discussed.

Reference has been made to the use of such hairs in the scientific investigation of crime.

Eight cases have been quoted to illustrate various points of interest in the examination of hairs from the head and other parts of the body.

I am indebted to Dr. J. B. Firth, *C.B.E.*, F.R.I.C., M.I.Chem.E., Director of the Home Office Laboratory, Preston, for permission to submit this article for publication.

I should like also to record my appreciation of the considerable assistance afforded me by Mr. W. Dickin (of the Biology Department) and Mr. H. Rowbottom (of the Photography Department) of the Laboratory in the preparation of the photographs.

MEDICO-LEGAL CASE BOOK

III. A Fabricated Shooting Incident

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Early one morning I was informed of the attempted burglary of a house on which there was a police guard. It was alleged that there had been an exchange of shots, one of which had passed through a guard's shirt without causing him any injury. Routine investigation included careful examination of the shirt, which was immediately photographed.

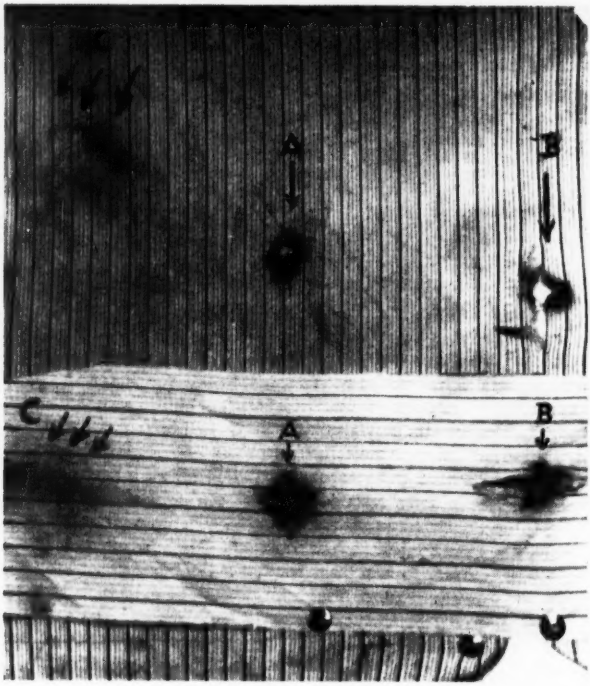

Fig. 1

I was convinced that the scientific evidence was incompatible with the story of the police guard who stated that, some time after midnight (he did not know exactly when) he disturbed a man trying to break into the house he was guarding. The intruder fled and the guard, in an attempt to cut off his escape, leapt over a wall at the end of a verandah

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in front of the house. While the guard's hands—his right hand was holding his revolver—were still on top of the wall, which was of about his own height, the intruder shot at him from a distance of 80 cm., and the shot penetrated the guard's shirt, though the guard himself was unscathed.

Examination of the shirt (Fig. 1, upper half) showed it to be of coat-

Fig. 2

style blue and white striped poplin, the neck band 38 cm. On the left-hand front panel there was a cross-shaped tear (A), 36 cm. from the point of the shoulder and 17 cm. from the centre edge. This tear measured 2.5 cm. vertically and 2 cm. horizontally. It was in the centre of a circular area of blackening. A second cross-shaped tear (B) lay about 12 cm. from tear A, to the rear of it on a slightly lower level. Tear B measured 5 cm. vertically and 2.5 cm. horizontally, with an area of blackening both inside and outside. A further area of blackening (C) lay to the front of tear A, about 9 cm. from it on a slightly higher level. Tears A and B were clearly of the type which would result from the firing of a pistol whose muzzle was in contact with or very close to the cloth.

The policeman's statement was so obviously false that the shooting incident as he had described it was reconstructed and photographed (Fig. 2). Next an experiment was carried out. Using the guard's own weapon and ammunition, a bullet was fired through a similar poplin shirt in the way we deduced the original shooting must have occurred.

A fold of material from the left side of the shirt was held in the left hand and the revolver was pressed against the fold with the right hand and fired (Fig. 3). The appearance of the resulting tears and blackening

Fig. 3

(Fig. 1, lower half) was identical with that of those on the guard's shirt. The blackening at C was due to the blow-back from the breech end of the weapon, and occurs only with revolvers and not with automatic pistols. In addition, chemical examination of the blackened areas on the guard's shirt showed that they had been caused by a powder of a similar composition to that contained in his ammunition.

These facts proved conclusively that the police guard's account of the shooting had been fabricated.

Exclusion of Parentage by Rh-Hr Blood Tests

A Modification of Boyd's Compact Tabular Presentation

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In 1950 one of us[1] published a table of matings in which the Rh–Hr phenotypes of the parents were given, the Rh–Hr types possible among their children, and the types of children which excluded parentage. In this table it was assumed that tests had been carried out with antisera anti-**Rh_0**, anti-**rh′**, anti-**rh″**, anti-**hr′**, and anti-**hr″** so that 18 different phenotypes could be distinguished. The resulting table of 171 matings covered a total of 6 closely printed pages.

About the same time, in the first edition of their book *Blood Groups in Man*, Race and Sanger[2] presented a table of Rh–Hr matings showing the types possible among the children for each mating. In this table it was assumed that tests had been carried out only with reagents anti-**Rh_0**, anti-**rh′**, anti-**rh″**, and anti-**hr′**, as is the standard current medico-legal practice. These matings were listed using the so-called short-hand notations (the original Rh–Hr notations with the omission of 'h'), but employing them as genotypes, while phenotypes were indicated by bracketing all the possible genotypes corresponding to the phenotype in question. With this cumbersome device, the resulting table of only 76 matings consumed a total of 20 pages.

In Keynes' book[3] on *Blood Transfusion* a third table of matings for the Rh–Hr types can be found. Here the Rh–Hr types are represented by the C–D–E genotypes, and again phenotypes are represented by bracketing all the genotypes corresponding to the phenotype in question. Here an even more complex table resulted which covers a total of 6 pages.

Recently Boyd[4] has shown how, by arranging the various matings in the form of a checkerboard, and by using a number code to indicate the phenotypes of the children, it is possible to show all the matings on a single wide page. This makes it a simple matter to find any desired mating for medico-legal purposes. In his table Boyd assumed that the tests had

Table 1: Exclusion of Parentage by the Rh–Hr Tests

Phenotype of Putative Mother	*Phenotype of Putative Father*				
	1 rh	2 rh′rh	3 rh′rh′	4 rh″	5 rh′rh″
1. rh ..	2, **3,** 4, 5, **6,** 7, 8, **9,** 10, 11, **12**	**3,** 4, 5, **6,** 7, 8, **9,** 10, 11, **12**	1, **3,** 4, 5, **6,** 7, 8, **9,** 10, 11, **12**	2, **3,** 5, **6,** 7, 8, **9,** 10, 11, **12**	**3, 6,** 7, 8, 10, 11, **12**
2. rh′rh ..	3, 4, 5, 6, 7, 8, 9, 10, 11, 12	4, 5, 6, 7, 8, 9, 10, 11, 12	1, 4, 5, 6, 7, 8, 9, 10, 11, 12	3, 6, 7, 8, 9, 10, 11, 12	7, 8, 9, 10, 12
3. rh′rh′ ..	**1,** 3, **4,** 5, 6, **7,** 8, 9, **10,** 11, 12	**1, 4,** 5, 6, **7,** 8, 9, **10,** 11, 12	**1,** 2, **4,** 5, 6, **7,** 8, 9, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 8, 9, **10,** 11, 12	**1, 4, 7,** 8, **10,** 11, 12
4. rh″ ..	2, **3,** 5, **6,** 7, 8, **9,** 10, 11, **12**	**3, 6,** 7, 8, **9,** 10, 11, **12**	1, **3,** 4, **6,** 7, 8, **9,** 10, 11, **12**	2, **3,** 5, **6,** 7, 8, **9,** 10, 11, **12**	**3, 6,** 7, 8, 10, 11, **12**
5. rh′rh″	3, 6, 7, 8, 9, 10, 11, 12	7, 8, 9, 10, 11, 12	1, 4, 7, 8, 9, 10, 11, 12	3, 6, 7, 8, 9, 10, 11, 12	7, 8, 9, 10, 12
6. rh_yrh'	**1,** 3, **4,** 6, **7,** 8, 9, **10,** 11, 12	**1, 4, 7,** 8, 9, **10,** 11, 12	**1,** 2, **4,** 5, **7,** 8, 9, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 8, 9, **10,** 11, 12	**1, 4, 7,** 8, **10,** 11, 12
7. Rh_0 ..	2, **3,** 4, 5, **6,** 8, **9,** 10, 11, **12**	**3,** 4, 5, **6, 9,** 10, 11, **12**	1, **3,** 4, 5, **6,** 7, **9,** 10, 11, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9,** 12
8. Rh_1rh..	3, 4, 5, 6, 9, 10, 11, 12	4, 5, 6, 10, 11, 12	1, 4, 5, 6, 7, 10, 11, 12	3, 6, 9, 12	none
9. Rh_1Rh_1	**1,** 3, **4,** 5, 6, **7,** 9, **10,** 11, 12	**1, 4,** 5, 6, **7 10,** 11, 12	**1,** 2, **4,** 5, 6, **7,** 8, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7,** 10
10. Rh_2 ..	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9,** 12
11. Rh_1Rh_2	3, 6, 9, 12	none	1, 4, 7, 10	3, 6, 9, 12	none
12. Rh_2Rh_1	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7,** 10

To understand and use this chart quickly and easily, the phenotype names and numbers should be u
in conjunction with the Table of Rh–Hr reactions.

1. Determine the phenotypes of the blood of the mother, putative father, and the child or chil
using potent and specific anti-**Rh_0**, anti-**rh′**, anti-**rh″**, and anti-**hr′** sera including appropriate contr
2. Assign the corresponding phenotype symbol and number to each individual tested.

Table 1: Exclusion of Parentage by the Rh–Hr Tests—*continued*

	Phenotype of Putative Father						
5 rh′rh″	6 rh_yrh'	7 Rh_o	8 Rh_1rh	9 Rh_1Rh_1	10 Rh_2	11 Rh_1Rh_2	12 Rh_2Rh_1
6, 7, 8, 11, **12**	**3,**4,**6,**7,8,**9,** 11, **12**	2, **3,** 4, 5, **6,** 8, **9,** 10, 11, **12**	**3,** 4, 5, **6, 9,** 10, 11, **12**	1, **3,** 4, 5, **6,** 7, **9,** 10, 11, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**
8, 9, 10,	4,7,8,9,10, 12	3, 4, 5, 6, 9, 10, 11, 12	4, 5, 6, 10, 11, 12	1, 4, 5, 6, 7, 10, **11, 12**	3, 6, 9, 12	none	1, 4, 7, 10
4, 7, 8, 11, 12	2, **4,** 5, **7,** 9, **10,** 11,	**1,** 3, **4,** 5, 6, **7,** 9, **10,** 11, 12	**1, 4,** 5, 6, **7,** **10,** 11, 12	**1,** 2, **4,** 5, 6, **7,** 8, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11
6, 7, 8, 11, **12**	3, 4, **6,** 7, 9, 10, 11,	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**
8, 9, 10,	4, 7, 8, 9, 11, 12	3, 6, 9, 12	none	1, 4, 7, 10	3, 6, 9, 12	none	1, 4, 7, 10
4, 7, 8, 11, 12	2, **4,** 5, **7,** 9, **10,** 11,	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11
6, 9, 12	3, 4, **6,** 7, 10, **12**	2, **3,** 4, 5, **6,** 8, **9,** 10, 11, **12**	**3,** 4, 5, **6, 9,** 10, 11, **12**	1, **3,** 4, 5, **6,** 7, **9,** 10, 11, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**
none	4, 7, 10	3, 4, 5, 6, 9, 10, 11, 12	4, 5, 6, 10, 11, 12	1, 4, 5, 6, 7, 10, 11, 12	3, 6, 9, 12	none	1, 4, 7, 10
4, 7, 10	2, **4,** 5, **7,** **10,** 11	**1,** 3, **4,** 5, 6, **7,** 9, **10,** 11, 12	**1, 4,** 5, 6, **7,** **10,** 11, 12	**1,** 2, **4,** 5, 6, **7,** 8, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11
6, 9, 12	3, 4, **6,** 7, 10, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**
none	4, 7, 10	3, 6, 9, 12	none	1, 4, 7, 10	3, 6, 9, 12	none	1, 4, 7, 10
4, 7, 10	2, **4,** 5, **7,** **10,** 11	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, 7 8, **10,** 11

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d or chil
riate cont

Find the assigned number of the mother and father on the side and top columns of the Table and locate the box at which both intersect.

The child or children are not possible from this mating if their assigned numbers appear in this box.

Phenotype numbers given in bold face represent children for whom maternity is excluded.

been carried out with the 4 most commonly used antisera, anti-$\mathbf{Rh_0}$, anti-**rh'**, anti-**rh"** and anti-**hr'** so that 12 phenotypes and 144 matings could be represented. Dr. Boyd kindly sent us his table before publication. After seeing his table we felt that although it represents a marked advantage over previously published tables, it does not use the form of presentation to maximum advantage. We have, therefore, prepared another table (Table 1) which utilizes Boyd's principle but differs in certain details, so that information becomes available that is not given in his table. As a result our table differs considerably from that published by Boyd.

1. The table of Boyd gives the CDE as well as the original Rh–Hr notations. This we consider to be a disadvantage. It is desirable that only a single uniform terminology be used for the Rh–Hr blood types just as for the other blood group systems. In our chart we use the original Rh–Hr nomenclature alone.

2. In presenting the CDE notations Boyd uses symbols which in part resemble phenotypes and are in part genotype symbols. This method of using the CDE symbols is used by no other worker in the field and, as a result, adds still another variation to the list of more than 20 CDE symbols for the identical Rh–Hr phenotype.

3. In his presentation of the Rh–Hr phenotypes Boyd uses his own novel variation differing from the standard. In the accepted Rh–Hr terminology the capital R is reserved for those phenotypes which are characterized by the presence of the $\mathbf{Rh_0}$ factor, while phenotypes in which the $\mathbf{Rh_0}$ factor is absent are represented by small r's.

Table 2: Reactions Characterizing the 12 Rh–Hr Phenotypes

Phenotypes	*Reaction with Sera*			
	Anti-$\mathbf{Rh_0}$	Anti-**rh'**	Anti-**rh"**	Anti-**hr'**
rh	−	−	−	+
rh'rh ..	−	+	−	+
rh'rh' ..	−	+	−	−
rh" ..	−	−	+	+
rh'rh" ..	−	+	+	+
rh_yrh' ..	−	+	+	−
Rh_0 ..	+	−	−	+
Rh_1rh ..	+	+	−	+
Rh_1Rh_1 ..	+	+	−	−
Rh_2 ..	+	−	+	+
Rh_1Rh_2 ..	+	+	+	+
Rh_ZRh_1 ..	+	+	+	−

4. In our table the phenotypes are arranged in a 'natural' order (rh, rh'rh, rh'rh', rh", rh'rh", etc.) which makes it easier to find a specific mating. (The serological reactions defining each of the 12 Rh–Hr phenotypes are given in Table 2.)

5. In Boyd's table the numbers of those children are omitted whose types would not be expected to occur because of incompatibilities with the mother's blood type. In our modification the types of the children which exclude maternity are listed but in bold face type, so that our table is more complete and informative.

6. In Boyd's table, column 11 is omitted because the only children excluded would be those whose blood types are incompatible with the type of the mother. Inclusion of this column makes our table more informative and less ambiguous, without any significant increase in size.

7. Boyd's table omits the column and row in which the mother and father belong to the rare type rh_yrh' because the gene r^y is so rare that it would hardly ever in practice play a part in any case. At the same time, however, he includes matings in which one or both parents belong to type rh′rh″, overlooking the fact that as many as half the individuals of this type are of genotype $r^{y}r$.

8. Because of its completeness and arrangement, our modification of Boyd's table possesses symmetry which the original table lacks.

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Blood Spot Identification

Experiences in the Police Laboratory

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The identification of blood spots is routine in a well-organized police department laboratory. As Director of the Police Laboratory of the St. Louis Metropolitan Police Department, I am especially charged with all serological procedures, of which blood spot identification plays the lead.

The following figures represent the serological examinations performed by us during the past 5 years: (1950), 357; (1951), 516; (1952), 447; (1953), 553; (1954), 536. Total—2,409. About half were blood stains; the other half were seminal stains.

All types of exhibits for the purpose of identification of blood spots are encountered in a police laboratory—shirts, handkerchiefs, other articles of wearing apparel, shoes, weapons, material scraped from furniture and from walls. In many cases, spots from automobiles are brought in, stains wiped from windshields, dried blood from automobile seats, blood spots from metal parts of the car where a fatal or near-fatal injury has occurred.

One of the most important aspects of blood stain examination is the preparation and procurement of blood specimens for identification purposes. I am much indebted to Dr. Alexander S. Wiener, who submitted a directive to the Police Department of New York on this question. His views and mine are incorporated in the following:

1. *State of Preservation of the Stains.* Bacteria will destroy the bood stains at body or room temperatures. A stain that is not completely dry on cloth that is rolled will often develop a mold. Direct personnel not to roll up any garment that is stained by fresh blood. Excessive heat and excessive cold will destroy a stain, the latter condition not being as destructive as the former. The ideal temperature is refrigerator temperature of approximately 5 ° C. or 41 ° F.

2. *Procedure.*

I. *Dried Stains.*

1. Remove the dry stain by carefully prying up some of the crust by means of a clean, dry scalpel or knife. Do not remove all of the stain from the area.

2. (a) If on a removable object, deliver the object, with the remaining stain, to the laboratory immediately, together with the removed dry crust, as found *in situ.*

(b) If on a non-removable object, on walls, floors, etc., wherein the blood has been absorbed, remove a representative unstained section as well as a section which

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supported the stain by gouging or digging out. These specimens are to be placed in separate clean containers and delivered to the laboratory as soon as possible, together with the dry crust. If the non-removable section is glass or a polished area where there was no absorption or penetration of the stain, no control area is absolutely needed.

II. *Moist Stains.*

1. Dry the stains quickly and completely by means of waving in the air or directing a fan on the area.
2. Handle the dried stain as described in I.

III. *Liquid Stains.*

1. Use a dry, clean dropper to take up some of the stain or use a clean, dry test tube to scoop up some of the stain. If taken up with a dropper or pipette, place it in a clean, dry test tube and seal.
2. Place the sealed test tube in a beaker or such container of cracked ice.
3. Completely and quickly dry the remainder of the stain by waving in the air or directing a fan on the area. Then handle as described in I.
4. Deliver the liquid and dry specimens to the Director of the Laboratory as soon as possible.

It is our custom to mark all exhibits on which blood spots are found with initials of the examiner (RBHG) on some prominent portion of the material. This is done with indelible pencil and large enough to be readily found when the exhibit goes to the courtroom. We also make it a point to mark with pins the places that have been examined, especially when we have cut pieces out from the material for blood spot tests.

Experts are not supposed to be concerned about esthetics. I must mention the fact, however, that when the exhibits are presented to the expert on the witness stand for identification he must first of all identify his own initials or whatever mark he has put on the exhibit. This necessitates handling exceedingly filthy articles of wearing apparel. Sometimes it is necessary to search for a while for the identifying marks. If one is on the witness stand and has had to identify say half a dozen or a dozen types of filthy garments, one necessarily has an overwhelming desire to wash one's hands as soon as possible. Courtrooms as a rule have no facilities for washing the hands, so one must be patient and wait until the examination is over before swiftly repairing to an adjacent washroom where this self-purification can take place.

There are 3 sources of blood concerned in these serological examinations in homicide cases:

1. Blood spots found at the scene of crime; also bloodspots found on the clothing of the accused; blood spots of exhibits such as automobiles, weapons, found at the scene of the crime or on the person or in the custody or in the possession of the accused;

2. A specimen of blood from the body of the deceased victim. This is usually obtained through the assistance of the coroner's autopsy physician, the coroner, or the medical examiner's autopsy physician. This should be received in a sterile bottle or test tube properly closed and submitted to one of the laboratory staff so that the chain of evidence will not be broken. In St. Louis we have a perfect

liaison with the coroner's autopsy physician, Dr. John Conner, who has always been very co-operative in obtaining the blood specimens properly and avoiding breaking the chain of evidence;

3. A blood specimen is taken from the person of the accused. Later we will discuss the question of the rights of the accused to refuse the examination, and the rights of the Police Department to obtain them.

Integrity of Chain of Evidence

One of the most important aspects of this as well as all other types of laboratory examination commonly performed in police laboratories is the *integrity of the chain of evidence.* This is most easily obtained by ensuring that all evidence is collected by members of the laboratory staff and no one else. This requires the laboratory staff to work in close harmony with the homicide squad. Material gathered by the laboratory staff is brought to the laboratory or to the Director's office, routinely entered in the laboratory book of entry with time, names, etc., all written and receipts properly given by those who receive the specimens. Full written data must be compiled covering who brought in the specimen, the time brought in and the date. A receipt should be given to the member of the personnel who brings in the material and that member should also make a written statement that he has brought in the material.

One must be prepared to submit all this written evidence during the trial of the case. We have for several years had no difficulty in court proceedings with this aspect of investigation, but we very well recall one case where the results of blood examinations were excluded because the chain of evidence was broken.

Some one in the Police Department, not a member of the laboratory staff, received a specimen of blood from the victim of an assault and was not able to identify the person from whom he had received the specimen. A young child had been kidnaped and assaulted and subjected to injuries, including a head injury, and was brought to a local hospital for operation on the skull. We requested a specimen of the victim's blood from the surgeon. A sergeant of police instead of one of our personnel was sent to the operating suite where he received from the hands of an unnamed nurse a vial of blood ostensibly from the victim. He neglected to open the package and look at it and identify it; he also neglected to get the name of the nurse from whom he had received it. The material was delivered to the District Commanding Officer instead of to the laboratory. In the trial of the case, my statements about this blood and its blood groups and types were objected to on the grounds that it could not be shown by evidence that the blood really came from the victim. It happened to be a case in which the liquid blood of the victim corresponded to the blood group found on the assailant's clothing; his own blood was of a different group. Thus it happened that a probably guilty man walked out of the court room as a free man because of negligence in preserving the integrity of the chain of evidence.

Rights of the Accused

There is another matter in connexion with this work which should be discussed, viz. the rights of an arrested or accused person who refuses to

permit his blood to be taken. Despite various court rulings it still remains a matter of doubt whether any police department is empowered to take a specimen of blood from an accused person without his permission. In our Department we do not take blood without the consent of the accused.

So far as we know, the State of New York is the only Commonwealth that has passed legislation covering the question of procurement of blood from an accused person. Mr. William E. Leonard, Acting Commissioner, State of New York, Department of Correction, has written to us:

'Section 940 of the Code of Criminal Procedure was added to the Code by Chapter 875 of the Laws of 1928, but authority for the taking of blood grouping tests was not vested in police officials until the enactment of Chapter 592, Laws of 1943, which became effective 1 September 1943.

'While Chapter 607, Laws of 1940, effective 1 September 1940, purported to amend Section 940 of the Criminal Code, and Sub-division 3 of Section 552 of the Criminal Code, the only text set out was that for Sub-division 3 of Section 552, and consequently there was no change in the blood grouping provision of the law. Also, Chapter 349, Laws of 1947, effective 25 March 1947, amended Section 940 of the Criminal Code, but likewise made no change in the Blood Grouping Provision.

'Section 940 of the Code of Criminal Procedure is effective at the present time.'

Section 940 (Identifying criminals; taking of finger-prints) reads:

'In order that the courts and public officials dealing with criminals may have accurate information as to the identity of persons charged with crime, there is hereby conferred and imposed upon the chief of police or peace officer performing such functions, in each city, town or village, and upon sheriffs, members of the state constabulary, the railway police, the aqueduct police, the state park police and all other police officers making arrests, the power and duty of causing to be taken upon arrest, fingerprints and thumbprints, and if necessary the photograph, and if necessary the blood grouping tests, of every person arrested and charged with a felony or with any of the misdemeanors and offenses specified in Section 552 of this code. For the purpose of maintaining the identity of such photographs an identificational numeral board or other similar apparatus or device may be used as required. Members of the state police, upon arresting a person or persons for any felony or any of the misdemeanors and offenses specified in Sec. 552 of this code may transport or bring said persons arrested to their troop headquarters for the purpose of fingerprinting and thumbprinting and photographing and, if necessary, blood grouping.'

In view of these facts it seems proper to suggest to the Academy here assembled that steps should be taken through one of its committees to see to it that this legislation is enacted in all other States. At the same time, while not quite *apropros* the subject of my paper, corrective legislation should be enacted concerning the question of the use of blood grouping tests in paternity cases. This also has been enacted into a law in the State of New York. For a full discussion of this question, I refer to Mr. Sidney B. Schatkin's Chapter 22 in *A Textbook on Legal Medicine** entitled *Paternity Proceedings and Blood Tests.*

* Edited by R. B. H. Gradwohl, The C. V. Mosby Company, 1954.

Problems in Identification of Blood Stains

With the arrival and proper receipt of the exhibits in the police laboratory, there are 3 problems which ought to be solved in relation to the identification of blood stains.

i. Is the stain produced by blood?

ii. Is it human blood?

iii. To what blood groups and types does it belong?

The first problem is best answered by the use of chemical tests, viz. the benzidine test and the phenolphthalin test. The phenolphthalin test is that recommended by Gettler and Kaye. We consider it the more reliable of the two. A third test for the identification of the spot as blood is the spectroscopic examination. This should always be done on all specimens because the first two tests mentioned are unspecific.

The test to determine whether or not the spot is human blood is the so-called ring or precipitin test. The discovery of this method of identification of blood was made almost simultaneously by Uhlenhuth, Wassermann and Schuetze. (7 November 1901, by Uhlenhuth, and a day later by Wassermann).

There was no thought at the time of its discovery of its use as a medicolegal procedure for the determination of blood spot identification. Originally the test was thought of as a means of scientific species identification. A classical account of blood relationships by means of the precipitin test will be found in Nuttall's book, *Blood Immunity and Blood Relationship* (1904). Nuttall was University Lecturer in Bacteriology and Preventive Medicine at Cambridge, England. Nuttall's interest was not primarily based upon the question of medicolegal features of the precipitin test for blood; rather was it his desire to show there was a common property in the bloods of certain groups of animals; the property had persisted throughout the ages which had elapsed during our possible evolution from a common ancestor. It was an early classical outline of an adventure in evolution, pointing the way so that others might follow.

Some time after its discovery the use of the precipitin test was suggested for medicolegal purposes in the identification of the blood of Man and its differentiation from that of other animals. Many years have elapsed since the date of the orginal discovery. Many changes in the technique of the preparation of antisera have been published. As a rule, most of the antisera are prepared in rabbits by the injection of dilute solutions of human blood plasma, intravenously, subcutaneously or intra-abdominally. The test has been developed to a point of great accuracy and has been adopted in most police laboratories for the identification of human blood. Reports have been submitted to courts with unqualified statements about the specificity of the test. Occasional early references were made to the

fact that some of the primates might give similar results with the anti-human precipitin serum, using the ring test technique.

After many years of deliberation, I finally decided to put this matter to a crucial test, viz. procurement of the blood of the primates in sufficient quantities to make anti-primate sera, checking this against human blood, and checking the primate's blood against anti-human precipitin serum. An Editorial appeared in the *South African Medical Journal,* 2 February 1952, making the following very pertinent remarks concerning the precipitin serum test for human blood, as follows:

'The field of forensic pathology is redolent of myth. Unverified allegations continue to be copied uncritically from textbook to textbook and almost literally handed down from father to son. The province of serology is no exception to this rule, and major unwarranted assumptions about the specificity of the tests for human blood continue to be made. Modern serologic techniques permit the revaluation of the adequacy of the dogmas based on an inadequate appreciation of the limitations of the routine laboratory . . .'

Before I read this editorial and the publication of the work of Dr. J. E. Duncan Taylor in the same issue of the *South African Medical Journal,* I had been at work for some months on the preparation of antisera against the bloods of chimpanzees, orangutangs and baboons. As a result of my efforts, I published in *The Laboratory Digest* (February 1952 and May 1952) my results using chimpanzee blood in the ring test against the anti-human precipitin serum and anti-chimpanzee serum against human blood and against chimpanzee blood. These results are set forth below.

The chimpanzees belong to the classification of the Pan-Chimpanzees. Previous results with the blood of other monkeys, *Macaca mulatta* and the blue-faced mandrils and other small baboons, were completely negative, i.e. monkey blood showed no resemblance in the ring test for human blood, using anti-human precipitin serum. Investigations were also made by me on gorilla, and orangutang blood, all showing the same results that I obtained with chimpanzee blood. Anti-human precipitin sera gave the same positive results with chimpanzee, gorilla, orangutang blood specimens as with actual samples of human blood. Likewise anti-chimpanzee serum precipitated chimpanzee, gorilla, and orangutang as well as human blood. Anti-gorilla sera precipitated gorilla and orangutang as well as chimpanzee and human blood. Anti-orangutang sera precipitated orangutang the same as gorilla, chimpanzee and human blood. My conclusion at the time of the publication in 1952 was that there was no simple practical method available for laboratory testing which can distinguish human blood stains from the blood stains of chimpanzee, gorilla or orangutang.

These facts are not especially important in many countries where chimpanzees, gorillas and orangutangs are not ordinarily found at the crime scene, but the exclusions of reactions with blood with these species is of considerable importance in certain countries, e.g., South Africa. There, the greater proportion of exhibits submitted for medicolegal examination is obtained in the cases of murder or rape, etc., affecting a non-European population, in particular, the Bantu. In many of the rural areas of that country, e.g., in Zululand, monkeys and 'bush-babies' are killed with

weapons such as assegais, which are also used in many cases of assault. Likewise, in other parts of the same country, the exhibits are likely to be contaminated with baboon blood. Furthermore, the dried skins of monkeys are frequently worn by the Bantu as ornaments or clothing.

Dr. Duncan Taylor reviewed the various methods of making anti-human precipitin serum as outlined in various textbooks of forensic medicine. By absorption methods, Taylor and Gordon believe it is possible so to absorb anti-human precipitin serum that it can be useful in differentiating between the reactions of human and various types of primate bloods. This technique was outlined to me by Prof. I. Gordon in a personal communication. While in no way disputing the accuracy of Gordon and Taylor's methods, my attempts to verify them have failed completely.

Another aspect for solution of this problem is found in the work of Gemeroy, Boyden and De Falco, from the Serological Museum, Bureau of Biological Research, Rutgers University, New Brunswick, N. J. Here, instead of the ring test, they have utilized the Libby Photronreflectometer. This instrument measures in terms of turbidity. With it the whole range of reaction of antigen-antibody mixtures may be measured directly in the cells in which they are mixed. It is objective and quantitative; a single test through the entire range of anti-serum dilutions can be run in about an hour. These investigators consider the flocculation test using the Libby Photronreflectometer a valuable adjunct in clarifying the differentiation between various mammalian bloods, including the differentiation between human and primate blood. We would recommend that police scientists fully investigate these claims.

In addition to the routine ring test technique, we use a number of controls beyond those ordinarily recommended. We use a normal saline control, a control of rabbit's blood, guinea pig, chicken, dog, beef and horse blood. We also routinely check the unknown blood with anti-chicken, anti-beef, anti-dog and anti-horse sera. Accused persons frequently try to account for the presence of blood on their garments by statements that the blood contaminated their garments from chickens which they had killed, or that it was blood from cattle, horses or dogs. By checking with all these antisera, these statements can easily be tested. If these antisera are not used, defendants' attorneys are likely to cast doubt on laboratory findings. We have found it most illuminating and useful to be able to refute these statements *in advance* rather than to leave them in doubt in the actual hearing of evidence. Counsel very properly may embarrass an expert witness by calling more attention to what he has *not* done than to what he *has* done.

The police laboratory must be sure that the anti-human serum used in any given case is reliable and potent. Antisera purchased on the market must be certified. Expiration dates must be watched, for these antisera

sometimes lose their potency by aging. We usually use several samples of anti-human sera of different dates of manufacture. We make all of our own anti-sera, anti-human, anti-dog, anti-chicken, anti-horse and anti-beef. In this way we have no misgivings about the reliability of the ring test. Our constant thought is a close scrutiny of all biological procedures. It must be remembered that these are not chemical tests, but tests of an entirely different nature. There cannot be too much care or too many methods of control.

After the chemical and serological tests for blood have been made, it is necessary to attempt a classification of the blood groups of the specimens. These blood groups are as follows: O, A, B and AB, and differentiation of A_1 from A_2. Next, attempts to demonstrate the types M and N and MN; then, attempts to find group Rh and sub-groups of Rh. The International groups O, A, B and AB are easily demonstrated in dry blood. The M agglutinogen holds up fairly well in dry blood, but the N factor disappears rapidly. We have found, too, that the Rh factor rapidly disappears in dry blood. Naturally, all the groups and types are easily found in the specimen of blood from the victim and from the accused. Evidence pointing to the type or the group of blood is very important in many cases. The blood group finding is often the most important part of the laboratory test in trials of these cases. It goes without saying that the greatest care and accuracy must be used in all the work of the police laboratory, especially so in connexion with biological procedures.

The expert, both in direct and cross-examination, must be very precise and clear in explaining his findings to the court. He must take care to give his results in clear, understandable English. By his manner, diction and demeanor he must indicate to the court his impartiality and present all the facts, both his own findings and the opinions of other experts, so that the utmost degree of fairness can be established in the minds of the jury.

At this point, it is to be emphasized that the blood tests used in homicide cases require expert knowledge. Blood grouping tests commonly performed for purposes of blood transfusions are not the same as those used in these investigations. Many laboratory technicians and directors of hospital laboratories are quite well informed on these tests as performed for purposes of safe blood transfusions, but they are singularly uninformed in many cases on the type of test used in an examination of dry blood spots. These require absorption methods which are definite enough, but must not be confused with the ordinary blood grouping and typing tests performed in the hospital laboratories. No one should attempt to perform these tests unless he is well informed about absorption techniques and interpretations. It is needless to remind this audience that often the findings of the serologist are a deciding factor in proving the guilt or innocence of the accused and in no case must an expert forget this very important consideration.

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Poisoning by Aspirin and Paraldehyde

P. R. v. d. R. Copeman, B.A., D.Sc.
Government Chemical Laboratories, Johannesburg

Among the synthetic drugs, used as analgesics, antipyretics and hypnotics, aspirin and paraldehyde may be classed with the barbiturates in regard to the wide appeal which they have for the general public. In the form of self-administration all these compounds are widely used for the relief of pain, low fever and insomnia. On this account and owing to the ease with which aspirin and preparations containing paraldehyde can be obtained, it is inevitable that cases of poisoning by these substances will occur through misadventure or mis-use. The fact that relatively large doses must be ingested to produce toxic symptoms induces a false sense of security, while the nature of the effects produced by these drugs makes them attractive to those who contemplate suicide.

The use of paraldehyde for inducing sleep was introduced about 1870, some 5 years after the introduction of chloral into general use for this purpose. Aspirin (acetyl salicylic acid) was introduced into use about 1900 in the hope that it would be converted slowly in the system into salicylic acid and thus avoid the irritant action of the free acid. Aspirin has proved to be the most successful drug as an analgesic for the milder types of pain and as an antipyretic for low fevers. In order to avoid confusion in discussing these two drugs they will be treated separately.

(1) Aspirin

The antipyretic and analgesic properties of salicylic acid have been used medicinally for a considerable period but it suffers from the disadvantage of producing toxic effects. The various efforts to modify this toxicity by using derivatives which would liberate salicylic acid in the system slowly, led to the introduction of acetyl salicylic acid. This compound has proved most successful. The drug has been marketed under a variety of trade names of which, perhaps, 'Aspirin' is the most widely known and has, in fact, been accepted by the B.P. as the synonym for acetyl salicylic acid. Aspirin has an action similar to that of salicylic acid, but does not exhibit the cumulative toxic action of the compound and large doses are required to produce toxic effects. According to Cushny,[3] it passes through the stomach with very little change and is free from the gastric effects of salicylic acid. However, it suffers some decomposition in the intestinal tract and thus the effects of aspirin ingestion may be due partly to the salicylic acid as well as the antipyretic effect of aspirin itself. In cases of poisoning, the products isolated from the viscera are cleavage products of which the most important is salicylic acid.

Owing to its analgesic properties and the ease with which large amounts of aspirin can be obtained, this drug has not infrequently been used for suicidal purposes. The therapeutic dose is given as 5–15 grains, but it is difficult to state with any exactness the amount of a fatal dose. Webster[7] states that the toxic dose of aspirin for adults is about 150 grains but, owing to susceptibility, fatal cases may arise from smaller doses. On the other hand, there has been recovery from larger doses.

During the period 1948–1952, 20 cases involving death from aspirin poisoning were examined in the Government Chemical Laboratories, Johannesburg. In addition there were 7 cases in which both aspirin and a second drug were involved; 12 of the 20 cases involved the deaths of adults and the remaining 8 cases were those of children, due apparently to overdosage. In this connexion, a communication to the *South African Medical Journal*[4] is of interest. It is stated that salicylate intoxication is encountered frequently in paediatric practice in Johannesburg and the warning is given that morbidity and mortality in infancy can only increase with the use of aspirin. Among the cases examined there was one case of the death of a Native child due to poisoning by methyl salicylate. This is not an unusual case as, before this period, cases of poisoning of European adults by methyl salicylate have been examined in this laboratory.

Methods of Investigation

Aspirin is an organic compound with weakly acid properties, and its method of isolation from the specimens submitted follows the same general course as has been described for barbiturates.[2] Owing to the decomposition which occurs during the process of extraction, aspirin appears as its decomposition product (salicylic acid) in the extract, which will contain the 'acidic' compounds originally present in the viscera. Suitable methods of purification are used to separate the salicylic acid in a pure form and the final residue is weighed. This residue may then be identified by various well-established tests for salicylic acid and the amount of salicylic found can be calculated in terms of aspirin.

The problem arises, of course, whether aspirin or salicylic acid has been ingested originally. From a practical point of view this is not of great importance, since the cases of death are all suicidal or accidental and, for the reasons given, cannot always be settled satisfactorily by a purely chemical examination. A positive decision whether aspirin or salicylic acid had been originally ingested must depend largely upon the previous history of the case. In some cases a detailed examination of the stomach contents may provide the necessary information in regard to the presence of aspirin, but this may not always be possible.

Distribution of Aspirin in the Body

Aspirin is a synthetic organic compound and does not occur normally in the body. Any salicylic acid formed in the system by the decomposition of aspirin will exercise its usual effects, but the undecomposed aspirin will produce its usual antipyretic effect. However, the distribution of aspirin in the system after ingestion will be essentially that of salicylic acid which.

after administration, is found in all the organs and secretions of the body. According to Cushny [3] salicylic acid appears in the urine within an hour of its adminsitration and is all eliminated in 48 hours. This conclusion does not appear to apply to aspirin. In one case, No. 4452/52, that of an Indian male child, 82.3 p.p.m. (mg. per kg.) were recovered from the liver and a total of 81.8 mg. from the organs submitted where death had occurred 3 days after the adminstration of aspirin. The brain is said to contain less than most of the other organs and, in Case No. 536/49, in which the brain was also submitted, only traces of salicylic acid could be detected in the brain while the liver, for example, contained 50 p.p.m. Symptoms of idiosyncrasy are more common with aspirin than with other derivatives of salicylic acid and thus fatal cases of poisoning may arise from relatively small doses of aspirin. Webster [7] states that in aspirin poisoning a marked acidosis may develop and that this is not common in poisoning directly with salicylic acid.

Death from Aspirin Poisoning

Although salicylic acid derivatives, such as aspirin, may be absorbed rapidly from the intestines, large doses are generally required to produce toxic effects. Despite this, fatal cases have arisen from aspirin poisoning. In some cases these may be accidental, due to overdosage, but cases have been examined which are definitely suicidal. Since large doses are required to produce toxic effects, it is to be expected that large amounts of aspirin will be found in the organs after death. Table I gives the average amounts in terms of parts per million (mg. per kg.) found in the organs submitted in cases of death from aspirin poisoning. Cases involving the deaths of children or cases where a second drug has also been involved have been excluded.

Table I: Death of Adults from Aspirin Poisoning

Organ	*No. of Cases*	*Average*	*Maximum*	*Minimum*	*Standard Deviation*	*Coefficient of Variation*
Liver (L) ..	11	248·8	1320·0	Trace	398·8	160·3
Kidneys (K)	11	231·0	975·0	Trace	296·6	128·4
Stomach ..	11	422·4	1928·1	Trace	618·0	146·3
Intestines ..	8	547·8	2549·0	Trace	—	—
R=L:K ..	11	0·963	1·36	0·39	0·332	24·4

The average concentrations found in the various organs are higher than those found for barbiturates and this appears to indicate a somewhat lower toxicity. In one case, No. 583/51, that of a European female, it was established that the deceased had taken 200 aspirin tablets (each presumably of 5 gr.). The concentration in the liver was found to be 1320 p.p.m. and a total of 6.14 gm. (=92.2 grains) was recovered from the liver, kidneys, stomach and intestines. This case is a good illustration of the difficulty of recovering a fatal dose even when a massive dose has been ingested. It

must be borne in mind that, apart from the destruction of the compound in the tissues, there was, in this case, vomiting which caused a further loss of the ingested aspirin. The problem of the amount of a drug originally ingested cannot be solved by a chemical examination.

The 'coefficient of variation' indicates how widely the amounts found in the organs may vary. Factors such as amounts taken, susceptibility and time of death will have a direct bearing on the amounts found, but these cannot be assessed. However, the value of R = L : K indicates the same tendency as in the case of barbiturates[2] for the concentrations in the liver and in the kidneys to become equal. The average value of this ratio was found to be 0.963, which does not differ significantly from unity. The concentrations in the stomach and intestines indicate that large doses have been ingested.

Death of Children

In view of the communication mentioned concerning the effects of aspirin administration in the case of children,[4] the results obtained in such cases are of interest. There were 8 cases of the death of children from aspirin poisoning during the period 1948–1952 and the data obtained have been summarized in Table II in terms of parts per million in the same way as in Table I.

Table II: Death of Children from Aspirin Poisoning

Organ	*No. of Cases*	*Average*	*Maximum*	*Minimum*	*Standard Deviation*	*Coefficient of Variation*
Liver (L) ..	8	66·3	174·0	Trace	54·5	82·2
Kidneys (K)	8	154·4	273·0	Trace	113·4	73·4
Stomach ..	8	161·9	433·3	Trace	148·2	91·5
Intestines ..	8	130·9	394·3	Trace	—	—
R=L:K ..	8	0·53	1·00	0·24	0·32	60·2

The information available indicated that these cases were all accidental, due to an overdosage or to an allergy. It will be noted that, as expected, the average concentrations are lower than those reported in Table I and thus support the contention that some danger is attached to the indiscriminate administration of aspirin to children. In addition, the distribution in the system of children seems to be different from that in adults, at any rate so far as the liver and kidneys are concerned. The difference in the concentration in the liver and the kidneys of children was found to be 88.1 p.p.m., with a 't' value of 2.75. For a 20 to 1 significance this should be 2.37[5] and the difference is therefore significant. This conclusion is supported by the average value of R = L : K, viz. 0.53, which differs significantly from unity, the 't' value of the difference being 4.16. In the case of children, therefore, the concentration of aspirin in the kidneys is definitely higher than in the liver. This may be due to a more efficient detoxication mechanism in the liver of children or possibly to a greater renal excretion.

Although the concentrations in the liver and kidneys of children are, on the average, lower than in adults, the individual variations in each group are so wide that no statistical significance can be shown in the respective differences between each group. However, the internal distribution in children does vary from that in adults as shown by the average values for R = L : K. The difference is 0.43 with a 't' value of 2.83 compared with 2.11 for a 20 to 1 significance. Thus, in the case of children, aspirin tends to accumulate to a greater extent in the kidneys than in the case of adults.

Aspirin and a Second Drug

Although it cannot be asserted that, in cases where a second drug had been ingested simultaneously, death was due to aspirin poisoning, the data obtained in such cases are of some interest, particularly in regard to the distribution of the aspirin in the system. The results obtained in 4 cases are given in Table III in terms of parts per million.

Table III: Aspirin with a Second Drug

Second Drug	*Liver (L)*	*Kidneys (K)*	*Stomach*	*Intestines*	*R = L : K*
Strychnine ..	62·9	25·0	420·1	—	2·51
Coramine ..	105·5	258·4	46·8	140·0	0·40
Barbiturate ..	44·0	62·8	44·2	156·5	0·70
Phenacetin ..	93·8	94·6	74·3	114·4	0·99

The results are insufficient for definite conclusions to be drawn but there are one or two points which are suggestive. Where strychnine had been ingested it will be noticed that the ratio (R = L : K) was high. Since strychnine is a very rapidly-acting poison, it would seem that death was sufficiently rapid to prevent much absorption of aspirin from the intestinal tract and that there was little opportunity for effective elimination through the kidneys. In the other cases the action of the drug is such that sufficient time elapses to allow a more normal distribution of aspirin in the system.

Diagnosis of Aspirin Poisoning

Individuals differ greatly in their reactions to the effects of salicylic acid derivatives and serious symptoms may be produced by relatively small doses. Symptoms of idiosyncrasy are more common with aspirin than with other derivatives. Because aspirin is probably more widely used than any other drug of this type, this peculiarity may be more apparent than real. Congestion of the nasal passages is commonly encountered in aspirin poisoning and this may be accompanied by signs of gastro-intestinal irritation. Nausea and vomiting may occur and marked acidosis has been reported from aspirin administration.[7] In the 27 cases examined during 1948–1952, vomiting was mentioned on 6 occasions while diarrhoea was mentioned on 2 occasions. In 2 cases the victim was in a comatose condition.

Webster[7] states that post-mortem appearances may include hyperaemia

of the stomach and intestines with congestion of the lungs and haemorrhages in the heart, lungs and meninges. The stomach and intestines may show the lesions of a true gastritis or enteritis. On 5 occasions out of 27 gastritis was mentioned as a sign of poisoning and congestion of the organs was described in 3 cases.

In most cases no cause of death was suggested, while in 4 cases salicylic acid poisoning was suggested. In 2 cases cantharidin poisoning was suggested. In 3 cases poisoning by paraldehyde, barbiturate and 'metals' was put forward respectively as the cause of death. In the remaining cases the conclusions were absent or at best indefinite.

It is clear from this summary that any decisions regarding the cause of death based solely on post-mortem appearances or even on symptoms are open to considerable doubt. A full history of the cases, together with the results of a chemical examination, is necessary before a correct diagnosis can be made, and all the available information should be at the disposal of the analyst before he commences his examination.

(2) Paraldehyde

Paraldehyde is a synthetic hypnotic which has enjoyed great popularity and is still frequently used, with the inevitable result that deaths from paraldehyde poisoning are by no means infrequent. It resembles alcohol in its general effects but it is a much more powerful narcotic and it rarely produces any symptoms of excitement. Paraldehyde is rapid in action but suffers from the disadvantages of having a pungent odour and taste and from having an irritating effect upon the mucous membranes. Although prompt in action, the duration of its effects is relatively short. For these reasons it has largely been supplanted by the barbiturates.

During the period 1948–1952, 18 cases of death involving paraldehyde were examined in the Government Chemical Laboratories, Johannesburg, and *all* were cases of suicide or of accidental poisoning. Of these only 2 cases involved the death of Natives. There were 3 cases in which barbiturates were also involved as a second drug while in 1 case arsenic was also present in the viscera. In this last case the deceased was stated to have taken two bottles of Somnex, a paraldehyde preparation, and one bottle of Antexit; 1514.5 mg. of paraldehyde and 36.3 mg. of arsenic were recovered from the liver, the kidneys, the stomach and the intestines in this particular case.

In comparing the incidence of poisoning by paraldehyde and by aspirin it must be borne in mind that paraldehyde is normally a liquid while aspirin is marketed in tablet form. In the former case overdosage is likely to occur while, in the latter case, tablets make a wider appeal to the general public. Aspirin probably has a wider use than any other synthetic drug.

Distribution of Paraldehyde in the Body

Paraldehyde is a polymer of acetaldehyde and traces of acetaldehyde may normally be present in the tissues, especially as a partial oxidation product of alcohol. The amount, however, is extremly small, since alcohol is normally oxidized to carbon dioxide and water in the system. A few cases have been investigated with this problem in mind. In those cases where no alcohol was present in the blood, no aldehyde could be detected and, where alcohol was present, only traces of aldehyde were found. In a paper dealing with the determination of acetaldehyde in blood, Stotz[6] states that the amount decreases in the blood on standing, even in an ice-chest, and ascribes this to cellular metabolism. He quotes various authorities to show that blood may contain normally from 0.22 to 0.37 p.p.m. of acetaldehyde. It is clear that a full history of a case is essential to enable correct conclusions to be drawn when only traces of aldehyde are present in the organs. Paraldehyde is excreted by the lungs and imparts a characteristic smell to the breath, but it is chiefly excreted in the urine. Large doses may cause nausea and vomiting while periods of unconsciousness may last for 24 hours or more. The compound becomes distributed throughout the system and there is a marked odour when the body cavities are opened. The amounts found in the viscera will, of course, depend upon the amounts ingested and the time elapsing before death. In one case, No. 6382/52, a European male, 1371.5 mg. were recovered from the liver and 312.00 mg. were found in the brain.

Method of Determination

When specimens of viscera, viz. the liver, the kidneys, the stomach and/or the intestines, are submitted to this laboratory for examination, the presence of paraldehyde may usually be detected by its smell. This is a most characteristic indication of the presence of paraldehyde. Smell forms only one of the routine tests carried out in the initial stages of the examination of the specimens submitted. The various organs are weighed separately and when this test is positive aliquot weights of each of the finely divided organs are steam-distilled from an aqueous suspension acidified with sulphuric acid. The presence of an aldehyde is confirmed in the distillate by testing a few drops with Schiff's reagent which turns a bright red colour in the presence of aldehydes. The distillation is continued until a few drops of the distillate produce no colour with this reagent. Confirmatory tests for acetaldehyde are applied and the amount in the distillate is determined by a suitable method which will depend upon the amount of aldehyde indicated by the preliminary tests.

Death from Paraldehyde Poisoning

It is difficult to state the fatal dose of paraldehyde since very large amounts have been ingested without serious consequences except prolonged unconsciousness. Tolerance to paraldehyde can be established rapidly so that large doses can be taken with comparatively slight effects; this has frequently led to habituation. The therapeutic dose may be as high as 10 gm. (=150 grain)[1] and consequently large amounts can be recovered

from the viscera in cases of paraldehyde poisoning. In Table IV a summary is given of 12 cases of poisoning examined during the period 1948–1952. The results are reported in terms of parts per million (mg. per kg. of organ) and exclude 4 cases where a second drug was present as well as 2 cases where paraldehyde had been administered for medical reasons.

Table IV: Death from Paraldehyde Poisoning

Organ	*No. of Cases*	*Average*	*Maximum*	*Minimum*	*Standard Deviation*	*Coefficient of Variation*
Liver (L) ..	12	516·9	1258·7	7·0	428·1	82·8
Kidneys (K)	12	401·8	860·8	7·8	341·6	85·0
Stomach ..	12	1260·5	5476·0	7·5	1729·4	137·2
Intestines ..	10	492·3	1760·0	9·0	—	—
R=L:K ..	12	1·29	2·38	0·85	0·424	33·0

It will be noted that, with the exception of the intestines, the average concentrations of paraldehyde are higher than those in the case of aspirin. This would indicate a lower toxicity for paraldehyde than for aspirin but, at the same time, a more rapid absorption from the intestines. Except in the case of the stomach, the variations in the concentrations are not as great as they are with aspirin, and might be regarded as indications of decreased individual susceptibility as compared with aspirin. In one case, No. 1069/52, a European male, the concentration in the stomach was 5746.0 p.p.m. while the intestines contained 492.6 p.p.m. A total of 2.6 gm. (=39 grains) was recovered from the organs submitted. In case No. 3048/51, a European male, the liver contained 1258.7 p.p.m. of paraldehyde and a total of 3.97 gm. (=59.6 grains) of paraldehyde was recovered from the organs. In both these cases it is clear that massive doses must have been ingested, but at the same time, less than the therapeutic dose was recovered.

The distribution of paraldehyde between the liver and the kidney is such that there was a higher concentration in the former. The average difference was 115.1 p.p.m. and, using the method of controlled comparisons, this difference had a 't' value of 2.52. For a 20 to 1 significance 't' should be at least 2.2. Thus the concentration in the liver was significantly higher than in the kidneys. This conclusion is borne out by the value of 1.29 for the ratio R=L:K which has a 't' value of 2.37 for the difference from unity. It would appear, therefore, that elimination from the kidneys takes place more rapidly than destruction of paraldehyde by the liver.

The Effect of a Second Poison

A few cases have been examined in which a second drug has also been found present in the viscera. The results for such cases have been tabulated in Table V in terms of parts per million.

With the exception of the last case, where the liver was received in a dried-out condition, the concentrations of paraldehyde are somewhat lower

Table V: Paraldehyde with a Second Poison

Second Poison	*Liver (L)*	*Kidneys (K)*	*Stomach*	*Intestines*	*R=L : K*
Arsenic ..	270·6	472·0	310·3	590·9	0·58
Barbiturate ..	56·1	69·3	52·0	—	0·81
Barbiturate ..	54·6	56·9	258·0	142·7	0·96
Barbiturate ..	1371·5	—	—	—	—

than those usually found in cases of paraldehyde poisoning. In the case of the barbiturates there is the possibility of synergism. The distribution appears to be slightly altered so that the kidneys contain a higher concentration of paraldehyde than does the liver. However, no definite conclusions can be drawn and it is unlikely that death was directly due to paraldehyde poisoning.

Diagnosis of Paraldehyde Poisoning

Paraldehyde resembles alcohol to some extent in its action but it is a more powerful hypnotic and rarely causes any excitement. Its action is quite rapid and unconsciousness appears promptly, lasting for several hours.[7] Of the 18 cases examined, coma was mentioned on only 3 occasions. Large doses may cause nausea and vomiting but this last symptom was mentioned in only 2 cases. Paraldehyde has an unpleasant odour which may remain in the breath for a considerable time.

Post-mortem examination reveals nothing characteristic, but there may be signs of gastric irritation with some haemorrhage. There may be some congestion of various organs. The most characteristic sign is the strong smell of paraldehyde in the body cavities and this property was mentioned in 12 of the cases examined. It is clear that this is the most important indication of paraldehyde poisoning, and on 8 occasions paraldehyde was suggested as the probable cause of death. In this respect paraldehyde probably presents fewer difficulties in diagnosis than most other poisons.

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